ISBN 1188–4169

Canada Communicable Disease Report

FAX

Vol . 23–11

Date of publication: 1 June 1997

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NOTICE TO SUBSCRIBERS

You will recall that, in December last year, you each received a form from the Canadian Medical Association regarding your subscription renewal for the *Canada Communicable Disease Report* (CCDR). The form explained that, beginning in 1997, there would be two subscription rates: base and premium.

The annual base subscription includes 24 bimonthly issues (eight pages each), an index, six Advisory Committee Statements (eight pages each) and the Notifiable Diseases Annual Summary supplement. The base subscription is currently $86.50, including GST for domestic subscribers, and US$105 for foreign subscribers.

The annual premium subscription includes the issues provided under the base subscription AND eight additional supplements for approximately 350 extra pages of information. These supplements will be devoted to infection control guidelines, surveillance summaries on selected diseases, proceedings of workshops and consensus conferences, and contingency plans for unusual or emerging communicable diseases. The premium subscription is currently $160.50, including GST for domestic subscribers, and US$175 for foreign subscribers.

Two supplements entitled "An Integrated Protocol to Manage Health Care Workers Exposed to Bloodborne Pathogens" and "Preventing the Transmission of Bloodborne Pathogens in Health Care and Public Service Settings" have already been published this year, and distributed to those with the premium subscription.

If you receive the CCDR through the base subscription, it is possible to purchase copies of the supplements, provided through the premium subscription, separately by contacting the **Subscription Administrator, Canadian Medical Association, P.O. Box 8650, Ottawa, ON, K1G 0G8; telephone (613) 731-8610, ext. 2028; fax (613) 523-0937**. The price will depend on the number of copies requested.

The small number of recipients of the CCDR currently receiving it free of charge from LCDC are registered for the *base subscription only*. If you wish to receive any of the additional eight supplements you must purchase these directly from the Canadian Medical Association as indicated above.

CASE REPORTS: INVASIVE *HAEMOPHILUS INFLUENZAE* TYPE A INFECTIONS

The widespread use of *Haemophilus influenzae* type b (Hib) vaccines has resulted in a dramatic decline in invasive Hib disease[1]. However, other strains of *H. influenzae* are capable of causing invasive disease. This report describes two recent cases due to *H. influenzae* type a.

Case 1: In February 1996, a previously well 2-month-old male Aboriginal infant was referred to the emergency department by the family physician. The infant presented with a 24-hour history of fever, poor appetite, and lethargy preceded by a 1-week history of a respiratory illness. On examination, he was lethargic and extremely irritable. The infant was febrile (39° C), had a discharging purulent ear, crepitations on the left side of the chest, and decreased peripheral perfusion. Investigation found an elevated white blood cell count (32.3 x 10^9/L) with 50% neutrophils, and hemoglobin of 106 g/L. The chest x-ray showed a patchy consolidation in both lower lobes and the right upper lobe consistent with a bilateral bronchopneumonia with left pleural effusion. Lumbar puncture showed 2+ white cells. Throat swab grew 3+ ß-hemolytic group A streptococcus. Blood and

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cerebrospinal fluid cultures grew *H. influenzae* type a, sensitive to ampicillin and cefotaxime. The infant was initially treated with intravenous(IV) ampicillin and cefotaxime; only the latter was continued once sensitivities became available. He responded well to treatment and was discharged following 11 days of IV antibiotics. Rifampin was given to the infant at discharge and to household members due to the presence of two children < 48 months of age.

Case 2: A previously well 17-month-old male Aboriginal infant presented to the emergency department with a painful, swollen, right foot. A chair had dropped on his foot the previous day. Three months prior to admission, he had had a culture-confirmed case of pertussis. In addition, he had only received one dose of DPT-Polio (Act-HIB™) vaccine.

On examination, the infant had a low-grade temperature (37.1° C axillae), appeared flushed but not acutely toxic, and was not able to bear weight on his right foot; he had difficulty walking. The dorsum of the right foot was swollen and red; the redness and swelling extended above the ankle. Both tympanic membranes were inflamed. The white blood cell count was 16.2 x 10^9/L. An x-ray and bone scan found no evidence of fracture or osteomyelitis. Blood culture grew *H. influenzae* type a. The infant was admitted with a diagnosis of cellulitis and bilateral otitis media. IV cefuroxime was started. He showed gradual improvement and was discharged on oral amoxicillin-clavulanate after 6 days of IV antibiotics. There were no other pre-school children in the household.

Discussion

Type b infections predominate in early childhood. Non-typable and non-b typable strains increase in relative frequency with increasing age[2-4]. Although disease caused by non-b strains is clinically indistinguishable from Hib disease, those infected with non-b strains have a higher frequency of underlying disorders[5]. However, there was no clinical evidence of underlying disorders in these infants.

Overall, non-b typable strains are rare; types e and f are the most frequent. In a large, US, six-state, laboratory surveillance study, type a disease was responsible for less than 1% of invasive *H. influenzae* cases among all age groups[2]. A previous review of type a disease was only able to retrieve 12 reported cases[6]. In several instances, the portal of entry seemed to be an inflamed or injured upper respiratory tract. Case 1 of this report experienced a preceding respiratory illness while Case 2 experienced skin trauma to an area of cellulitis a day prior to presentation.

The two cases reported here involved Aboriginal children. While type a disease has been quite rare in population-based studies of invasive *H. influenzae* cases in the United States[2] and the United Kingdom[7], much higher rates of type a disease have been reported in Aboriginal populations in Australia[8] and Papua New Guinea[9]. A 3-year review of all invasive *H. influenzae* infections in pre-schoolers in northern Australia found that 8% of cases in Australian Aboriginal children were due to type a infection compared to none in non-Aboriginal children. A report from Goroka, Papua New Guinea, found that the type a strain was responsible for 12% of all *H. influenzae* meningitis in children, second only to type b. The reasons for this difference in epidemiology are unclear, although socioeconomic factors have been shown to increase the risk of invasive Hib disease[10]. Social conditions which promote earlier and repeated exposures to *H. influenzae* and other infections may lead to prolonged and high carriage rates[11] increasing the risk of invasive disease[9,12].

The introduction of Hib vaccine and the subsequent decline in Hib disease has raised the concern that other strains will emerge to cause invasive disease[13,14]. While carriage rates of Hib have diminished, there has been no apparent alteration in carriage of other strains[15,16]. In addition, while a cluster of disease due to types e and f has been described in a population following the introduction of Hib vaccine[14], no change in rates of non-typable and non-b strains have been identified at a population level[2].

It is still unclear what impact, if any, the reduction in carriage and rates of invasive disease due to Hib will have on non-typable and non-b strains of *H. influenzae*. Current reporting systems only capture type b infections. Therefore, sources of data, such as case reports, laboratory surveillance studies, and the results of typing of all invasive cases of *H. influenzae* must be relied upon to monitor the epidemiology of this disease.

In this report, type a strains were clearly shown to be associated with invasive disease in previously well children. In the first case, the household contained two children < 4 years of age and local public-health staff were faced with the question of whether to offer chemoprophylaxis to the household. It is not known whether non-b typable strains pose risks to young household contacts similar to type b strains. From a research perspective, information on carriage of these strains as well as the risk of development of invasive disease in household contacts would be useful, particularly if an increase in incidence of these organisms occurs.

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Editorial Comment

Haemophilus influenzae is a gram-negative bacterium which causes a variety of childhood illnesses, ranging in severity from relatively mild upper respiratory tract infections to serious invasive infections, such as meningitis and septicemia. *H. influenzae* strains may be either encapsulated or non-encapsulated. There are six antigenically distinct capsular serotypes, designated a through f. Serotype b accounts for > 95% of systemic *H. influenzae* infections in children. Non-typeable or non-encapsulated *H. influenzae* is a cause of pneumonia in children and adults, especially among the elderly and persons with chronic lung diseases. It is estimated to cause approximately 5% of invasive *H. influenzae* disease. Encapsulated serotypes, other than type b, have been known to cause invasive disease; however, their occurrence is relatively rare. There has been little information published regarding *H. influenzae* type a.

H. influenzae type b (Hib) is a nationally notifiable disease; a confirmed case is defined as laboratory isolation of Hib from a normally sterile site or the epiglottis, with symptoms clinically compatible with invasive disease. Invasive *H. influenzae*, other than serotype b, is not currently under national surveillance; consequently, the incidence of infection due to non-encapsulated *H. influenzae* or non-type b encapsulated *H. influenzae* is unknown.

Before the introduction of universal infant immunization in Canada, Hib was the most common cause of bacterial meningitis in children. The incidence of invasive Hib infection has declined dramatically since that time, from 2.6 per 100,000 population in 1988 to 0.2 per 100,000 in 1994. Whether immunization will result in an increase in infections due to non-encapsulated *H. influenzae* and non-b encapsulated serotypes is unknown. Case series and reports, such as the one by Moloughney et al, may provide an early indication of changing disease patterns.

Announcements

INTERNATIONAL CONFERENCES ON EMERGING INFECTIOUS DISEASES

The Center for Disease Control and Prevention and its partners are cosponsoring the International Conference on emerging Infectious Diseases (ICEID) 8 to 12 March 1998, in Atlanta. The purposes of the conference are to 1) exchange scientific and public health information about global emerging infectious disease issues, 2) present programs and activities that address emerging infectious diseases, 3) identify program gaps, 4) increase awareness in the public health and scientific communities of emerging infectious disease issues, and 5) enhance partnerships to address emerging infectious diseases.

The 4th International Conference on HFRS and Hantaviruses will precede the ICEID, convening 5 to 7 March 1988, in Atlanta. The conference will encourage exchange of scientific information about hantaviruses. Attendees may register for one or coregister for both conferences.

The call for abstracts and registration information will be available on the W3 at http://www.cdc.gov/ncidod/ncid.htm and published in the *Emerging Infectious Disease Journal* and other professional publications.

LABORATORY DIAGNOSIS OF GROUP A STREPTOCOCCAL INFECTIONS
New WHO Publication

This manual provides a detailed, expert guide to virtually all procedures commonly required for the laboratory evaluation of group A streptococcal infections. Noting the serious problems created by the resurgence of these infections and their severe sequelae, the manual aims to maximize the support that laboratories can provide at all stages of clinical diagnosis and treatment, research, and the management of epidemics.

The authors provide step-by-step instructions for the exact performance of over 60 laboratory methods. These range from basic culture techniques, through methods for serologic evaluation,

to highly sophisticated procedures for studying immune responses. Details of protocols that have been in use for more than four decades are complemented by brief descriptions of molecular and other non-serologic techniques for characterization of the organisms, making the manual suitable for use at peripheral laboratories as well as advanced research institutes. Of particular practical value is the inclusion of techniques for the production of typing sera, many of which cannot be purchased commercially.

Throughout the manual, advice on the basic rules of good laboratory practice, alerts to common pitfalls, and tips for good results draw their authority from the authors' extensive experience at two leading WHO Collaborating Centres for Reference and Research on Streptococci. Recommendations concerning the best or preferred methods are supported by thorough explanations, with particular attention given to procedures and precautions that can help reduce errors and improve the accuracy of results. More than 150 references to the literature are included in this comprehensive and authoritative guide.

The manual, which has 14 chapters, opens with an overview of the challenge posed by group A streptococcal infections and their sequelae, including rheumatic fever, rheumatic heart disease, acute post-streptococcal glomerulonephritis, and severe invasive group A streptococcal infections. The structure and antigenic composition of group A streptococci are also briefly reviewed. Subsequent chapters describe procedures for specimen collection and transportation, and explain how to perform basic techniques for bacteriologic culture, recognition, and storage.

Serologic techniques are covered in six chapters, which explain procedures for the determination of serologic group, characterization of strain, determination of T-protein agglutination patterns, and serotyping using several different methods. Streptococcal antibody tests are described in four chapters devoted to techniques for the determination of antistreptolysin O and antideoxyribonuclease B, the production of streptococcal antihyaluronidase, and the measurement of anti-M antibodies. The manual concludes with detailed practical instructions for the production of antisera, including several which must be prepared in the laboratory.

Intended for use by clinicians, technologists, research scientists, and epidemiologists, the manual should contribute to improving the precision and standardization of diagnostic work in industrialized and developing countries alike.

This publication can be obtained from the **Publications Department, Canadian Public Health Association, 400-1565 Carling Avenue, Ottawa, ON, K1Z 8R1, telephone: (613) 725-3769**). Price per copy is $36.92 (including postage, handling and GST).

Notifiable Diseases Summary

We have excluded this table from the electronic issue of Canada Communicable Disease Report for those readers who do not need this information. For those readers interested in this table, call the FAX*link* (1-613-941-3900 from a fax machine) and select the index to get the access number.

Notifiable Diseases Summaries published to date in the electronic format (FAX*link*) can be found in the index under the same name.

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